

PAPEL DA DISBIOSE INTESTINAL NO METABOLISMO DO TRIPTOFANO EM INDIVÍDUOS COM TEA

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RESUMO

Intestinal dysbiosis has been associated with systemic alterations that extend beyond the gastrointestinal tract, particularly in the modulation of the gut-brain axis. In the context of Autism Spectrum Disorder (ASD), recent evidence indicates that changes in the intestinal microbiota can impact essential metabolic pathways, especially tryptophan metabolism. This amino acid is a precursor of neuroactive metabolites involved in the regulation of mood, behavior, and neurological development. This study aims to analyze the relationship between intestinal dysbiosis, alterations in tryptophan metabolism, and their neuroimmune and behavioral implications in individuals with ASD. This is a narrative literature review based on recent studies addressing this interaction. Studies investigating metabolic, immunological, and structural changes associated with dysbiosis, as well as microbiota-targeted therapeutic interventions, were included. Findings indicate that intestinal dysbiosis in individuals with ASD is associated with a shift in tryptophan metabolism toward the kynurenine pathway, reducing serotonin production. This imbalance contributes to increased pro-inflammatory metabolites and oxidative stress, directly influencing clinical manifestations of the disorder. Additionally, microbial metabolites such as short-chain fatty acids may impair intestinal permeability and blood-brain barrier integrity, promoting neuroinflammation. Chronic low-grade immune activation was also observed, intensifying neuroimmune and behavioral alterations. Therapeutic strategies including probiotics, prebiotics, synbiotics, dietary interventions, and fecal microbiota transplantation show potential benefits, although further standardization is required. In conclusion, intestinal dysbiosis plays a significant role in ASD pathophysiology through modulation of tryptophan metabolism, highlighting new perspectives for innovative therapeutic approaches.

Palavras-chave: Dysbiosis; Intestinal; AND; Tryptophan; Metabolism.